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ORIGINAL RESEARCH

Predictors exploration of abnormal brain magnetic resonance versus normal brain computed tomography imaging in acute carbon monoxide poisoning

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Abstract

Acute brain lesions observed on magnetic resonance imaging (MRI) performed during acute-phase carbon monoxide (CO) poisoning were associated with patient prognosis. However, it may be difficult in critically ill patients because of the long examination time and for patients who have economical limitations due to the high price. The purpose of this study was to identify predictive factors for abnormal brain lesions on MRI in cases of normal brain findings on brain computed tomography (CT) in acute CO poisoning patients. This study was retrospectively analyzed at the tertiary emergency medical center located in Gyeonggi-do, Korea by prospectively collecting CO poisoning registry. From August 2016 to August 2019, 287 patients visited the hospital due to CO poisoning. Exclusion criteria included age under 18 years, being discharged against medical advice, no initial MRI data, no initial CT data, and having abnormal lesions on brain CT. Of the 103 patients included in the final study, the median age was 39 years old; 35 (34.0%) were male and 74 (71.8%) were intentionally exposed to CO. 27 (26.2%) patients had abnormal MRI findings. Based on multivariable analysis, elevated blood urea nitrogen (BUN) concentration (odds ratio, 1.165; 95% confidence interval, 1.037–1.308; p = 0.01) showed a significant association with abnormal MRI findings. The area under the curve was 0.753 (95% confidence interval, 0.636–0.869) in the receiver operating characteristic curve of BUN concentration for abnormal brain MRI presentations. Brain injury may be detected on brain MRI in acute CO poisoning patients even there was a normal brain CT scan. Our study revealed that elevated BUN concentration may be significantly correlated with abnormal MRI findings.

Keywords

Blood urea nitrogen; Carbon monoxide poisoning; Computed tomography; Hypoxic ischemic encephalopathy; Magnetic resonance imaging

1. Introduction

Carbon monoxide (CO) poisoning is a global health problem. In the United States, about 50,000 people visit the emergency department (ED) due to CO poisoning each year [1]. CO is a colorless, tasteless, and odorless toxic gas produced by incomplete combustion of carbon-based fuel and material [2]. CO binds to hemoglobin with a strength of about 250 times greater than that of oxygen; therefore, even with a small amount of CO, the oxyhemoglobin dissociation curve is shifted to the left [3]. Through this mechanism, CO can easily damage the brain via hypoxia by lowering the oxygen-carrying capacity [4].

In recent studies, acute brain lesions observed on magnetic resonance imaging (MRI) performed during acute-phase CO poisoning were associated with patient prognosis [5, 6]. However, MRI is more expensive and takes more time than computed tomography (CT). Where medical resources are insufficient, such as in developing countries, patients may have to be transferred to other hospitals for MRI. This could worsen the patient's condition during transfer and burden patients and their proxies with increased medical expenses. In addition, patients who cannot cooperate with the MRI because of its long examination time need to be sedated, which has risks of hypoxia, hypotension, and even cardiorespiratory arrest [7]. Therefore, it is important to decide which patients should undergo MRI.

Brain CT has excellent accessibility because it is cheaper and faster than MRI, but it is less sensitive for visualizing damaged brain lesions in CO poisoning patients than MRI [8]. Brain CT is commonly used before MRI to identify brain lesions in CO poisoning patients.

The purpose of this study was to identify predictive factors for abnormal brain lesions on MRI in cases of normal brain

findings on brain CT in acute CO poisoning patients.

2. Methods

2.1 Study design

This study was conducted using the CO registry with all CO poisoning patients who visited the tertiary emergency medical center located in Gyeonggi-do, Korea. The CO registry was prospectively collected in our hospital since 2016. The hospital institutional trial review board reviewed and approved this study.

2.2 Participation selection

From August 2016 to August 2019, we included in our study patients who had the proper history or physical signs after CO poisoning, and/or measured carboxyhemoglobin (COHb) values >5% in non-smokers (>10% in smokers) upon the initial presentation to the ED. Patients were excluded if they were under 18 years old, were discharged against medical advice, had no initial MRI data, had no initial CT data, and had abnormal lesions on brain CT (Fig. 1). According to our management protocol, brain CT was performed within 2 h after the presentation to the hospital and MRI scans within 24 h. However, imaging tests were not performed when patients or their proxies did not agree or when patients' medical conditions were included in the contraindication of imaging tests.

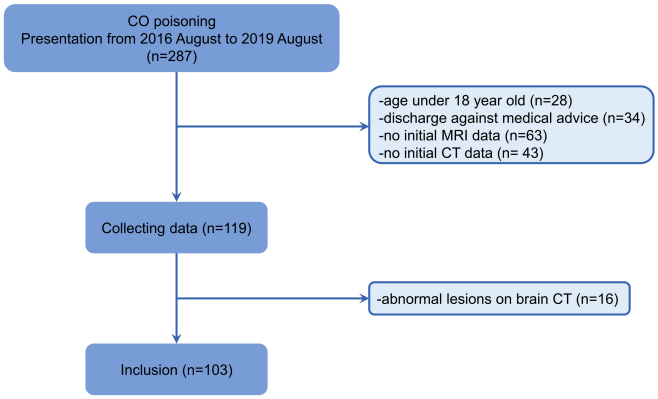

FIGURE 1. Flow chart of patient selection. Abbreviations: CO, carbon monoxide; MRI, magnetic resonance imaging; CT, computed tomography.

2.3 Data collection

Demographic data, laboratory results, symptoms, vital signs, medical comorbidities, Glasgow coma scale (GCS), intentionality of poisoning, and smoking status were collected for our registry at the ED visit. A CT scanner (Somatom Scope power; Siemens Healthineers, Erlangen, Germany) and MRI using a 3-T MRI unit (Signa HDXT 3.0; GE Healthcare, Chicago, IL, USA) were used. All brain images were reviewed by two blinded neuro-radiologists. Patients with any high signal intensity lesions on MRI diffusion-weighted images (DWIs) and low signal intensity on corresponding apparent diffusion coefficient (ADC) maps were classified into the abnormal MRI group. And patients with no high signal intensity lesions on DWIs or low signal intensity on ADC maps were classified into the normal MRI group. Electronic medical records were reviewed to prospectively collect patient laboratory results and brain imaging readings by two research nurses.

2.4 Management of CO poisoning

All CO poisoning patients received 15 L of oxygen per minute using a non-rebreather mask in the ED before delivering hyperbaric oxygen therapy (HBOT). HBOT was performed if the patient has following criteria: initial COHb ≥25% (COHb ≥15% in pregnant women); presence of neurological abnormalities such as seizure, loss of consciousness or altered mental status regardless of COHb concentration; signs of myocardial injury such as elevation of troponin I or abnormal electrocardiogram. HBOT was applied in a monochamber (IBEX MONO; IBEX medical systems Co., Tel Aviv, Israel). According to our hospital protocol, HBOT was conducted in three sessions within 24 h. For the first chamber session, the target pressure was 3 atm (absolute) and the total duration of HBOT was 150 min/session. For later sessions, they were planned for 2 atm and 120 min/session [9]. Compression was performed in the first 30 min of each session and decompression was performed 30 min before the end of each session.

2.5 Statistical analysis

SPSS Statistics for Windows version 26 (IBM, Armonk, NY, USA) was used for the statistical analyses. Continuous variables are presented as medians with interquartile ranges and categorical variables are expressed as absolute numbers or relative frequencies. Continuous variables that followed the normal distribution were compared using Student's *t*-test and those that did not were compared using the Mann–Whitney U test. The Pearson χ^2 test and Fisher's exact test were used for categorical variables. Multivariable logistic regression analysis was used to identify factors related to the results. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. And p-value <0.05 was regarded statistically significant, and all p-values were two-tailed. By using the Youden index, the receiver operating characteristic (ROC) curve was used to determine the optimal cuff-off value of the predictive factor (statistically significant predictor for abnormal MRI findings). The results showed a 95% CI of area under curve (AUC), sensitivity, specificity, positive predictive value and negative predictive value.

3. Results

A total of 287 patients visited our ED due to acute CO poisoning. Of these, 184 were excluded and 103 patients were ultimately included in our study. Those excluded were as follows: 28 were under 18 years of age, 34 were discharged against medical advice, 63 had no initial MRI data, 43 had no initial CT data, and 16 had abnormal lesions on brain CT (Fig. 1). All 16 patients with abnormal findings on brain CT showed abnormal MRI findings.

The median age of all patients was 39 years old and 35 (34.0%) were male. The median value of the initial GCS

was 14, 54 (52.4%) were current smokers, and 74 (71.8%) patients were intentionally exposed to CO. Loss of consciousness (25.2%) was the most common symptom, followed by headache (11.7%) and dizziness (9.7%). The median values of laboratory findings were as follows: neutrophil 76.7%, blood urea nitrogen (BUN) 15.1 mg/dL, creatinine 1 mg/dL, creatine kinase 123 U/L, and myoglobin 47 ng/mL.

Among the CO poisoning patients with normal CT findings, 27 (26.2%) patients had abnormal MRI findings. The median age of the abnormal MRI group was 44, which was higher than the normal MRI group with a median age of 37. The abnormal MRI group had an initial GCS score of 13, which was lower than the normal MRI group, and 3 (11.1%) patients complained of headache differently from the normal MRI group. Information on basic demographic data, vital signs, medical comorbidities, smoking history, GCS, and laboratory findings can be found in Table 1.

Univariable analysis revealed significant differences in laboratory findings, such as myoglobin, creatine kinase myocardial band, creatine kinase (CK), BUN, neutrophil, and creatinine ($p < 0.05$). CK (OR, 1.000; 95% CI, 1.000–1.000; $p = 0.01$), BUN (OR, 1.206; 95% CI, 1.097–1.325; $p < 0.001$), neutrophil (OR, 1.054; 95% CI, 1.011–1.100; $p = 0.02$), and creatinine (OR, 5.570; 95% CI, 1.136–27.315; $p = 0.03$). In addition, multivariable logistic regression analysis showed that BUN (OR, 1.165; 95% CI, 1.037–1.308; $p = 0.01$) was significant (Table 2). The AUC was 0.753 (95% CI, 0.636–0.869) in the ROC curve of BUN concentration for abnormal brain MRI presentations (Fig. 2). The cut-off value was 17.75 mg/dL with a sensitivity of 59.3% and a specificity of 85.5%. The positive predictive value was 59.3% and the negative predictive value was 85.5%.

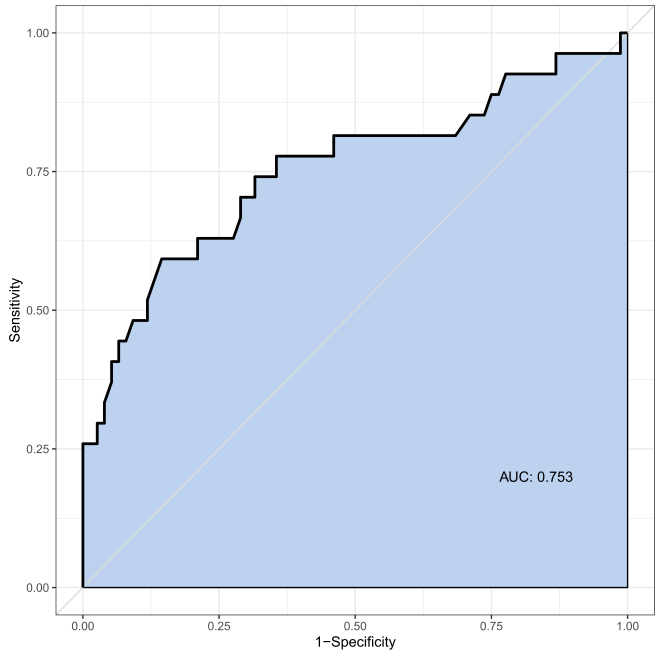

FIGURE 2. Receiver operating characteristic curves for predicting abnormal MRI finding according to blood urea nitrogen. Abbreviations: MRI, magnetic resonance imaging.

4. Discussion

In this report, we explored predictors of abnormal MRI findings with normal CT findings in acute CO poisoning patients. It is clinically important to determine when MRI, which is more expensive and takes more time than CT, should be performed. In this study, 27 (26.2%) patients showed abnormal MRI findings in CO poisoning patients with normal CT findings, and elevated BUN concentration was found to be a strong predictor of abnormal MRI findings (OR, 1.165, $p = 0.01$).

When hypoxic-ischemic encephalopathy occurs due to CO poisoning, not only acute neurologic symptoms but also late brain dysfunction such as personality changes, parkinsonism, and cognitive impairment can occur [10]. Brain imaging that directly evaluates structural damage would be more effective in predicting neurological deficits caused by CO poisoning than systematic factors such as laboratory findings [6]. Abnormal brain lesions on CT and MRI can be seen in CO poisoning patients because the brain is vulnerable to hypoxic injury [11, 12]. MRI is a more sensitive examination than CT and abnormal brain lesions caused by CO poisoning are seen by MRI as early as 1 h after exposure [12, 13].

According to a recent study, low GCS score and troponin I are predictors of acute brain lesions on MRI in CO poisoning patients [14]. However, there is no significant difference between the low GCS score and troponin I in our study. Our findings may have differed from those of the previous study because our study was conducted only in patients with normal CT findings. And people who attempted suicide through CO poisoning may have impaired consciousness by co-ingesting drugs or alcohol [15]. Additionally, patients who attempted suicide are often uncooperative with treatment, so it may be difficult to take their history [16]. Therefore, GCS can be affected by factors other than CO. In our study, 74 (71.8%) patients admitted for CO poisoning were intentionally exposed.

Troponin I is a diagnostic marker for evaluating myocardial injury in CO poisoning, but can also be used as a predictor for acute brain lesions on MRI because elevated troponin I suggests reduced blood supply to the brain, which can lead to neurological deficits [17, 18]. But our findings are not consistent with findings in previous studies. Because patients with moderate to severe damage as seen on brain CT were excluded, it was considered that troponin I might not be increased in either group in our study.

Unlike the United States, the number of suicide patients by CO poisoning is increasing in East/Southeast Asia, and the suicide attempt rate is inversely proportional to the socioeconomic status [19–21]. Acute brain lesions on MRI undertaken during acute-phase CO poisoning were associated with patient prognosis and delayed neurological sequelae [5, 6]. However, MRI is not always available for patients who live in areas with poor medical support, such as developing countries, or who are uncooperative or unstable. Therefore, it is important to identify predictive factors associated with abnormal brain lesions on MRI when the previously performed CT is normal.

In our study, BUN was significant as a factor to perform MRI. The BUN elevation caused by CO poisoning can be explained by several mechanisms. First, during CO poisoning,

TABLE 1. Comparison of baseline characteristics between normal MRI and abnormal MRI group.

	Total	Normal MRI	Abnormal MRI	*p*-value
	(N = 103)	(N = 76)	(N = 27)	
Age, year	39 [26.5–51]	37 [24.75–49.25]	44 [36–52]	0.09
Male, n (%)	35 (34.0)	27 (35.5)	8 (29.6)	0.75
BMI, kg/m^2	23.4 [20.4–26.3]	23.2 [20.3–26.2]	24.0 [20.8–26.5]	0.76
Vital signs				
Systolic BP, mmHg	129.0 ± 19.0	129.4 ± 18.9	128.2 ± 19.6	0.78
Diastolic BP, mmHg	80 [70–90]	80 [70–90]	80 [80–81]	0.90
Heart rate, BPM	93.1 ± 18.0	93.7 ± 18.8	91.6 ± 16.1	0.59
Respiratory rate, BPM	20 [18.5–20]	20 [18.75–20]	20 [18.5–20]	0.88
O_2 saturation, %	98 [96–98]	98 [95–98]	98 [96–98]	0.65
Comorbidities, n (%)				
Hypertension	9 (8.7)	8 (10.5)	1 (3.7)	0.44
Diabetes mellitus	2 (1.9)	1 (1.3)	1 (3.7)	0.46
Current smoking, n (%)	54 (52.4)	39 (51.3)	15 (55.6)	0.88
Initial GCS	14 [10–15]	15 [10–15]	13 [10–15]	0.26
Intentional exposure, n (%)	74 (71.8)	54 (71.1)	20 (74.1)	0.96
Symptoms, n (%)				
Headache	12 (11.7)	12 (15.8)	3 (11.1)	0.75
Loss of consciousness	26 (25.2)	19 (25.0)	7 (25.9)	>0.99
Dizziness	10 (9.7)	7 (9.2)	3 (11.1)	0.72
Chest pain	2 (1.9)	1 (1.3)	1 (3.7)	0.46
Laboratory findings				
WBC, 10^3/μL	12.1 [8.6–15.4]	11.9 [7.6–15.2]	13.1 [11.3–17.7]	0.10
Neutrophil, %	76.7 [64.2–84.0]	73.4 [63.3–83.3]	79.7 [76.9–85.5]	0.01
ANC, 10^3/μL	9.9 [5.3–13.0]	8.4 [5.0–12.8]	10.7 [9.0–14.0]	0.03
Hemoglobin, g/dL	14.8 [13.9–15.8]	14.85 [13.62–15.95]	14.6 [14.05–15.4]	0.50
BUN, mg/dL	15.1 ± 6.2	13.4 ± 4.2	19.8 ± 8.3	<0.001
Creatinine, mg/dL	1 [0.9–1.2]	1 [0.9–1.1]	1.1 [0.9–1.4]	0.04
Creatine kinase, U/L	123 [90–388.5]	120 [87–244]	191 [96.3–2813.8]	0.04
pH	7.4 [7.4–7.4]	7.4 [7.4–7.4]	7.4 [7.4–7.4]	0.97
Carboxyhemoglobin, %	9.9 [4.7–20.6]	10.2 [4.3–20.4]	7.8 [4.9–19.8]	0.70
CRP, mg/dL	0.1 [0.1–0.4]	0.1 [0.04–0.3]	0.2 [0.1–3.1]	0.11
Lactate, mg/dL	2.0 [1.5–4.1]	2.1 [1.4–4.0]	2.0 [1.6–3.8]	0.82
Myoglobin, ng/mL	47 [0.5–204.5]	28 [0–115.5]	154 [50–870]	<0.001
Troponin I, ng/mL	0.1 [0.1–0.2]	0.1 [0.1–0.1]	0.1 [0.1–0.3]	0.33
CK-MB, ng/mL	2.8 [1.4–8.6]	2.3 [1.4–5.0]	5.6 [2.1–13.8]	0.05
Receiving HBOT, n (%)	96 (93.2)	71 (93.4)	25 (92.6)	>0.99

Values are expressed as the mean ± standard deviation, median [interquartile range], or number (proportion). Abbreviations: MRI, magnetic resonance imaging; BMI, body mass index; BP, blood pressure; GCS, Glasgow coma scale; WBC, white blood cell; ANC, absolute neutrophil count; BUN, blood urea nitrogen; CRP, C-reactive protein; CK-MB, creatine kinase myocardial band; HBOT, hyperbaric oxygen therapy.

lipid peroxidation is increased due to the toxic effect of vascular endothelium, causing damage to blood vessels, which can lead to acute kidney injury (AKI) due to ischemia–reperfusion injury [22]. Also, rhabdomyolysis, which is a common complication of CO poisoning, can proceed to AKI due to the direct nephrotoxic effect of myoglobin, tubular obstruction and renal vasoconstriction [22, 23]. In our study, myoglobin was significantly elevated in patients with abnormal MRI. Deterioration

TABLE 2. Factors associated with the abnormal MRI findings accompanying normal brain CT findings.

	Univariable analysis		Multivariable analysis	
	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
Myoglobin, ng/mL	1.000 (1.000–1.000)	0.10		
CK-MB, ng/mL	1.020 (0.999–1.040)	0.15		
CK, ng/mL	1.000 (1.000–1.000)	0.01	1.000 (1.000–1.000)	0.49
BUN, mg/dL	1.206 (1.097–1.325)	<0.001	1.165 (1.037–1.308)	0.01
Neutrophil, %	1.054 (1.011–1.100)	0.02	1.047 (0.998–1.097)	0.06
Creatinine, mg/dL	5.570 (1.136–27.315)	0.03	1.920 (0.541–6.814)	0.31

Abbreviations: MRI, magnetic resonance imaging; CT, computed tomography; OR, odds ratio; CI, confidence interval; CK-MB, creatine kinase myocardial band; CK, creatine kinase; BUN, blood urea nitrogen.

of renal function caused by AKI may lead to the retention of nitrogen waste products and impaired control of electrolyte and extracellular volume [24]. Second, CO poisoning can lead to myocardial injury via hypoxia. This can affect cardiac output, use of angiotensin-converting enzyme inhibitors and diuretics, and neurohormonal vasoconstrictor systems, inducing renal dysfunction and increasing BUN concentration [25]. Third, serum BUN is one of the predictors of dehydration in patients with normal renal function [26]. Patients with CO poisoning may become dehydrated because there is no way to replenish water until they receive treatment, so BUN concentration may be an indicator of the time from the beginning of CO exposure to arrival at the hospital [27]. To our knowledge, there have been no studies reporting factors that predict abnormal MRI findings when CT findings are normal in acute CO poisoning patients.

This study had several limitations. First, since this was a single-center study, a larger prospective study is required to obtain more reliable results. Second, since CT and MRI were not performed at the same time, brain injury may have progressed between these time intervals. Third, our study result showed low BUN concentration difference between two groups and relatively low OR of BUN. However, considering standard deviation, the BUN concentration is 13.4 $\pm$ 4.2 and 19.8 $\pm$ 8.3 respectively; we can notice that the BUN value in the abnormal MRI group can exceed the upper limit or normal range of BUN concentration (BUN reference range, 6–20 mg/dL) [28]. In addition, since we only enrolled patients with normal CT findings by excluding patients with abnormal brain CT (e.g., hypoxic brain injury), severe CO poisoning patients might be excluded, so BUN concentration may not differ significantly. Fourth, this study revealed that elevated BUN concentration is related to abnormal brain MRI, so it is indirectly associated with neurological injury. However, the relationship between BUN elevation and other complications due to CO poisoning has not been studied. These limitations should be addressed in future multicenter studies.

5. Conclusions

Brain injury may be detected on brain MRI in acute CO poisoning patients even there was a normal brain CT scan. Our study revealed that elevated BUN concentration may be significantly correlated with abnormal MRI findings.

AUTHOR CONTRIBUTIONS

SH and YHL designed the study. SC, JL and JEM performed statistical analyses and drafted figures and tables. SN wrote the first version of the manuscript. SH and YHL approved the final version of the paper and edited it. All authors contributed to the final version of the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study protocol was approved by the Institutional Review Board of Soonchunhyang university hospital and conducted in accordance with the provisions of the Declaration of Helsinki (IRB file No. 2020-03-01). Review Board approved waiver of informed consent.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY

The data used to support the findings of this study are available from the corresponding author upon request.

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